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JACKSONIAN EPILEPSY: ADENOMA OF LIVER; ACUTE ASCITES WITH TUBERCLE BACILLI.

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OF NEW YORK.

A. A. was born August 7, 1885, and died January 16, 1897. Her father was always in good health, her mother at one period of her life neurotic. Father's family was without any morbid taint; mother's ancestors died when seventy-five, eighty-five, and ninety years old. She is the youngest of ten boys and girls, none of whom died young. One sister died of cerebro-spinal meningitis, one of "fistula" after having been insane, one brother (the oldest in the family) was an army officer during the civil war and died insane. Of the seven now alive one, a man of great abstract learning and a college professor, with healthy children, is epileptic.

A. A. weighed eight pounds at birth, sixteen when six weeks, twenty-eight when ten months old. Her first teeth (lower incisors) appeared at six months; the others, both temporary and permanent, came in due order and without any trouble. She walked when a year old. She was wet-nursed, never perspired on the head, lost no hair, had no thickened epiphyses, her limbs were straight. The only symptom reminding of rickets was constipation, which appeared at the age of six or eight weeks, and required enemata until she was five years old. At three years of age she had whooping-cough; at four, measles; no other disease.

In September, 1891, she fell from a swing, hurting the left side of her head. She did not get pale nor vomit; she cried, and appeared uninjured. In January or February, 1892, her right hand was noticed to twitch a little. In November she began to take music lessons, the twitching became stronger and more frequent, and the lessons were discontinued. On the first of November, 1893, she had on waking in the morning the first hard attack of convulsions, probably with loss of consciousness. The second attack of the kind took place on the 16th. There was complete loss of consciousness and slow recovery. Thick, incoherent talk; slight paralysis of the right arm. During two days previously the child, who was remarkably observing and intelligent, had noticed fatigue and pain in her right arm, and told a playmate she was going to have an attack. On November 24th she complained of tiredness and numbness in both arms, more so in the left. She slept poorly during that night, and experienced slight twitchings in her right hand. Feeling

an attack coming on at 6.30 A.M., she jumped up with the idea of preventing it. It was slight, with no loss of consciousness. Slight stiffness of legs. She said she had to hold on to the foot of her bed, as she was not quite steady. Afterward she felt absolutely well, and played all day. Had taken bromides two days; they were continued in moderate, and later on in larger doses, so as to give rise to bromism repeatedly. Slight twitchings of the right arm in the night of February 18, 1894. March 9th, while running, she tripped and fell, striking her right arm heavily. She complained at once of twitchings, which, though slight, continued over an hour. She was put to bed, but she complained of their coming more frequently while she was lying down; tying a cord round the arm above the elbow made the twitchings stronger. She seemed very nervous and worried each time the arm twitched. When she was read to the shaking stopped instantly, and she was well all day. Evidently this attack had but partly the significance of the former ones. Right arm shook badly on waking April 4th, after she had been playing, skating, and falling several times the previous afternoon; again on April 13th and May 22d. During two days previously she had complained of fatigue and pain of her left hand. She had been writing a good deal, however, with her left hand previously, having been taught and accustomed to use her left hand in place of the right when the local convulsions began. Slight attack on the 27th, two on June 2d.

Most of the attacks would come on in the morning on waking up; they lasted mostly a few minutes, and were but rarely attended with unconsciousness. She would often laugh and joke immediately afterward. During most of the attacks, and often days before, the child was pale; sometimes she was irritable before or after them, in some instances for days. But very few times she had more severe attacks during her sleep, breathing heavily and shaking all over her body. Before she was taken to Europe in the summer of 1894, she had, however, only very few of these hard attacks. Up to this time, and also later, she enjoyed the occasional advice of Dr. S. Weir Mitchell, who saw her both in Philadelphia and in New York. Drs. Gowers and Jackson saw her in London. It was principally the former who objected strenuously to the performance of an operation the advisability of which had been suggested.

During her European trip she was not changed. Slight attacks would come mainly when she was, or had been, fatigued; five during August, 1894. After three slight ones, on her return, she had a fully developed epileptic attack in her sleep on October 26th. After a few slight ones, in January, 1895, the attacks ceased entirely while she was taking, first fifty, afterward sixty grains of bromides daily, together with lithia and valerianate of zinc. The bromides having disagreeable effects (as heaviness, drowsiness, bloated face), were discontinued June 8th, and valerianate of zinc only administered. Attacks returned—one on the 25th, two the 26th, three the 27th, four the 28th, six the 29th, four the 30th (wetted the bed the first time), six on July 1st (was given again bromides, grs. 60 daily, zinc valerian., 30 grs., atropia,

gr. $\frac{1}{120}$), six on the 2d and on the 3d, nine on the 4th and on the 5th, five on the 6th, and three on the 7th. The bromides again showing a disagreeable effect, they were diminished, and the daily medication consisted of 30 grains of the bromides, 15 grains of valerianate of zinc, 10 grains of zinc oxide, and $\frac{1}{50}$ grain of atropia.

No attacks until December 21st (slight) and 22d (hard), after which she was given 60–70 grains of bromides daily, and 30 grains of valerianate of zinc, for one week. They were then stopped awhile; the attending physician, who saw her often every day, changing the medication as circumstances appeared to demand.

During 1896 the case changed for the worse. There was a severe convulsion on January 2d; it began in the right arm, and was mostly confined to the right side. Unconsciousness lasted but a few minutes, but there were great weakness and numbness of the right arm. It is quite important to remark that her intellectual faculties suffered very much less than the arm from an attack. On the 3d there was a severe local convulsion of the right arm without any unconsciousness, and one slight attack on the 15th, 16th, 18th (two attacks), and the 20th. On the previous night her pallor was excessive and her pulse feeble. In February she had twenty-one attacks of shaking or twitching of the right arm (one of the fingers of the right hand only) on ten days altogether. During March, 1896, the attacks, all of them localized, no general convulsions, became both more numerous and more severe. She had four on the 1st, five on the 2d, three on the 3d, 4th, 5th each, four on the 6th and 7th each, six on the 8th and 9th each, seven on the 10th, eleven on the 11th, fourteen on the 12th, twenty-seven on the 13th, twenty-six on the 14th, twenty-nine on the 15th, forty-nine on the 16th, twenty-five in the night of the 17th, twenty-six on the 18th between 7 A.M. and 9 P.M., twenty-eight on the 19th during the same hours, and nineteen in the following night. The last few days many of these local convulsions were attended with heavy breathing, the body was often drawn to the left while the right arm was drawn up or shaking, or both, and on the 19th the mouth was noticed to be drawn to the left.

It was on that day that Dr. Charles A. Dana saw the patient. To his kindness is due the following report:

"The patient was seen by me March 19, 1896. She was a well-grown girl for her years, and apparently well-nourished, but quite anæmic. Her mind was particularly bright and mature. She had a slight weakness of the right arm, but no paresis of the facial muscles or of the legs. The knee-jerks on both sides were normal, not exaggerated on the right side. The right arm showed a certain amount of ataxia. There was an inability to place the finger easily on the tip of the nose with the eyes closed, or perform delicate muscular movements, such as buttoning the clothes or picking up small objects from the table. There was also a slight lack of localization sense, so that she could not determine exactly the point touched by the hand or arm. There was, however, absolutely no anæsthesia, tactile, pathic, or temperature

anæsthesia. The elbow-jerk was present. There was no atrophy or any tremor or spasmodic movement in the arm. The tongue protruded straight, and there was no asymmetry of the facial muscles. The patient had no concentric limitation of the visual field or aural field, no loss of sense of smell or taste, no pharyngeal anæsthesia; in fact, absolutely none of the stigmata of hysteria. There was simply the weakness of the arm, with ataxia and defect in "motor touch," such as is found in disease of the motor cortex. During the examination she had several slight convulsive attacks; the arm was extended, the forearm pronated, the hand flexed, and the whole arm raised out somewhat from the side. The head was drawn over, to some extent, to the same side; but the eyes did not move. There was some twitching of the muscles of the face of both sides. There was no biting of the tongue, and had not been. The attack came on suddenly, and without cry or distinct aura; the patient asserted that it was not accompanied by loss of consciousness. It lasted only one or two minutes. After it was over the arm was, for a time, almost paralyzed, but after a few hours gained considerable strength. She would have a number of these attacks during the day. Shortly after my visits began they ceased, however, and the arm grew gradually stronger, less clumsy, and eventually she could use it nearly as well as the left, though never quite the same.

"The attacks were quite typical examples of a Jacksonian epilepsy. She had had at no time any severe headaches, no vomiting, and had no optic neuritis. The diagnosis at the time was Jacksonian epilepsy, due probably to some slight degenerative changes in a limited area of the motor cortex."

Under the direction of Dr. Dana she took on March 20th nitroglycerin, gr. $\frac{1}{200}$, and urethan, grs. 5, every two hours, bromides, grs. 50, through the day. She had thirteen severe attacks through the day (they had since becoming so numerous been more frequent during the day), and nine in the night; on the 21st forty-five and fifteen (bromides grs. 45, urethan grs. 45, nitroglycerin 6 tablets of $\frac{1}{200}$ gr.); on the 22d thirteen hard attacks and thirty-three slight twitchings from 6 A.M. to 10 P.M.; six times the right leg shook with the arm. There were four slight attacks in the following night (medication, bromides grs. 35, urethan grs. 50, tablets No. 7). On the 23d there were forty-nine slight twitchings between 8.30 A.M. and 10 P.M.; none in the night. Medication, bromides grs. 30, urethan grs. 55, tablets No. 3. The child was much brighter and stronger. On the 24th there were twenty-one slight twitchings between 7.30 A.M. and 9.30 P.M. Medication, bromides grs. 30, urethan grs. 60. On March 25th and the following days the same medication and no attack whatsoever at any time. On April 1st albuminuria was noticed, and urethan was given in doses of 45 grains; on the 3d less albumin, urethan 30 grains. This dose was continued. On the 6th there was hardly a trace of albumin, and the appetite good. At the same time daily doses of 30 grains of bromide were given; from July 5th to July 11th she took 25, after that 20 grains daily.

It should be here remarked that during the time of the greatest number and severity of the attacks large doses of bromides, up to 100 or even 120 grains,

were given daily, together with, or without, the valerianate of zinc, and had often to be discontinued because of serious bromism. The only time in which the *attacks ceased and the patient felt better at the same time was when she took urethan.*

She continued this medication while in the Catskill Mountains all summer, 1896. Her general condition did not improve; she lost flesh and was pale, though in fairly good spirits most of the time. The amount of albumin in her urine, which was considerable while she was having her attacks, diminished. But while there was hardly a trace and sometimes none in the middle of the summer, there was constantly a small quantity of bile in the urine. In October I saw her. She was in bed, pale (she *never was icteric*), with anorexia and a frequent pulse. She improved somewhat until in November she felt better, took drives, and with slight support once walked ten blocks. Then her general condition, however, changed for the worse, without any fever or other tangible symptoms. I saw her with Dr. Dana on December 1st. During that month she grew thinner and paler. On December 28th her nurse noticed some swelling of the abdomen, which had been rather sunk and lean. That swelling was observed plainly by the mother and by Dr. Dana on January 2, 1897. It was considered to be due to obstruction of the bowels; a high enema brought away a peculiar substance, mixed with some blood, of greenish color and offensive odor. The temperature was but slightly raised, and there was no pain. Within one or two days the abdominal cavity filled up with fluid, the diaphragm was somewhat impeded in its motion, respirations increased to thirty and thirty-six. No anasarca, no local œdema anywhere. The abdominal veins were very numerous and dilated, but no caput Medusæ.

A few days afterward her (moderate) dyspnœa became a little less annoying; the abdomen appeared a little less tense, and the veins somewhat smaller. Still this apparent temporary improvement did not last, and in a very few days the general condition suggested the necessity of an operation the beginning of which was to be a laparotomy. Dr. McBurney joined Dr. Dana and me in a consultation. The suddenness of the abdominal effusion suggested the presence of a thrombosis of the portal vein, or the presence of a tumor of some kind, perhaps tuberculous lymph-bodies, compressing the portal vein. The spleen had been made out to be large the previous week; the liver appeared somewhat swollen. Some solid or semi-solid masses could now and then be felt, but we could not be certain whether we had to deal with normal or inflamed and adherent intestines or neoplastic growths, or peritonitic exudations. During all this time her strength gave way slowly, with very poor appetite and a urine which, after having been copious, became scanty and dark. In the last few weeks its specific gravity ranged from 1025 to 1033, its urea about 0.031 in 1 c.cm. There was a faint trace of albumin, no sugar, little oxalate of lime, very little bile, a large amount of urates and phosphates, some bladder epithelium, and some little pus and mucus.

Laparotomy was performed by Dr. McBurney on the 10th. There was a

gallon or more of a thin, yellowish fluid in the abdominal cavity. Its specific gravity was 1011; it contained but little albumin and few salts. These examinations were made of specimens of fluid which were removed when the discharge was about half finished. The spleen was found to be large, the liver of moderate size, there were no adhesions between the intestines or between them and the abdominal wall or the viscera. No large neoplasms. The omentum was in good condition, the mesenteric glands were not swollen; the glands surrounding the portal veins were felt to be enlarged, so as to form a thick mass encircling and pressing on the portal vein.

The surface of the liver was shining and covered with very numerous yellowish-gray bodies. They were of different sizes, from the head of a pin to that of a bean. Those of the latter size were felt reaching far down into the liver tissue. They were taken to be tuberculous.

Many specimens of the abdominal fluids were centrifuged and stained, and examined for tubercle bacilli or other microbes. One in six such specimens yielded *large quantities of bacilli tuberculosis* in close proximity to each other.

The general condition of the patient did not improve after the operation. Her axillary temperature was never over 100.8° in the axilla, usually 100° or less, down to 98.8°; the respiration remained between thirty and thirty-six; her pulse rose until on the day of her death, January 16th, it reached 158, but little influenced by stimulant subcutaneous injections, which were continued until about twelve hours before she died.

The autopsy was made on January 17, 1897, twenty-four hours after death, by Dr. James Ewing. The following report is mostly from his pen. By his painstaking investigations extending over many weeks he has placed me under lasting obligations.

Report of Autopsy.—Body that of a moderately well nourished, distinctly anæmic child. Rigor mortis slight. No œdema or jaundice.

Heart. Pericardium normal. Right chambers moderately distended with clotted blood. Left chambers contracted, nearly empty. Valves, muscle, endocardium normal. No dilatation or hypertrophy.

Lungs. Show considerable venous congestion and œdema. Bronchial nodes deeply pigmented, not tuberculous. Pleural cavities contain a few drachms of serous fluid.

Peritoneum. Intestinal walls uniformly anæmic, peritoneal coat is slightly dull and in places granular. In pelvic cavity, about drainage-tube, are a few drops of pus, and a light purulent coating over adjacent coils of intestine. Parietal peritoneum shows evidences of intense venous congestion, presenting large patches of superficial dark and bloody infiltration. No miliary tubercles were anywhere seen, after close scrutiny.

Liver. Size about normal; surface and section dotted with very numerous light yellow nodules, from pin-head to pea-sized, very sharply outlined from the surrounding tissue. Some of these nodules project slightly above peritoneal surface. They are not caseous, but otherwise closely resemble miliary tubercles. There are many less on the surface than in the interior. The

hepatic tissue shows very distinctly the gross appearances of chronic congestion, the centres of the lobules being very deep red and depressed, the peripheries very light colored, but no blood oozes from the sections.

Gall-bladder. Contents and mucosa appear normal.

Spleen. Considerably enlarged, four to five inches long, consistence firm. On section the Malpighian bodies appeared very prominently set off from the deeply congested pulp tissue.

Kidneys show moderate venous congestion, but are otherwise normal. Adrenals normal. Pancreas normal. There is marked superficial congestion of ovaries, otherwise the genital organs are normal.

Intestinal mucous membrane appears normal. The solitary follicles are only faintly visible. The stomach is moderately congested and coated with mucus.

On opening the peritoneal cavity attention was at once directed to the condition of the portal vein. The gastro-hepatic omentum was found much thickened and very firm, the enlargement proving on dissection to be due to swelling of the periportal lymph nodes, four or five of which, measuring from 5 to 1 cm. in diameter, formed a nearly continuous mass along the portal vein. Undoubtedly these nodes during life produced considerable narrowing of the lumen of the vein. On removing the liver and a portion of the diaphragm, an enlarged lymph node, measuring $1 \times 1\frac{1}{2}$ cm., was found lying immediately above and adherent to the hepatic vein at its junction with the vena cava. It was hard, and its capsule covered with large veins, so as to look almost angiomatous. It did not seem possible that the hepatic vein could have escaped partial compression by this considerable mass of tissue. On section these lymph nodes appeared deeply congested, but not tubercular. No thrombi were found in either the portal or hepatic veins. The mesenteric nodes were not enlarged.

Brain rather large, the convolutions not flattened. Pia mater was opaque, whitish, thickened, and rather hard over a large surface; moderately congested, mostly so over the region of the motor centres. This change was more perceptible on the left side than on the right. In this neighborhood the convolutions were rather separated from each other and felt hard. This condition was suggestive of interstitial swelling in the white substance. The structure of the brain appeared normal everywhere, the ventricles were empty, the plexuses normal.

The left half of the *cranium* was smaller and flatter than the right.

Over the left motor centre there was a defect in the structure of the bone, $1\frac{1}{4}$ cm. wide and $2\frac{1}{4}$ cm. long, in a forward and downward direction. The bone was translucent, its outer surface quite smooth and on a level with the rest of the cranium, its inner surface depressed. This depression was quite sharp, as in craniotabic defects. There were two more such defects on the same left side, in a forward direction, quite as wide but shorter, about 3 cm. from the median line. One more such spot was found over the left brow; another was located over the left motor centre, $1\frac{1}{2}$ cm. long and $1\frac{1}{2}$ cm. wide. The space of the large fontanelle, $2\frac{1}{2}$ by 3 cm., was occupied by hard, thick, and rather irregular

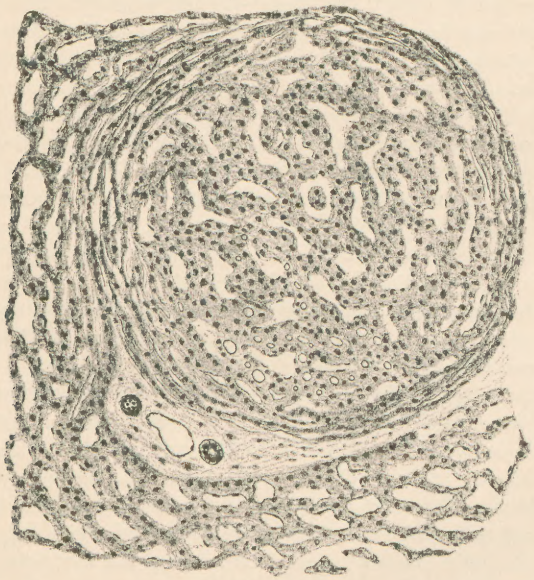
bone, which was surrounded by a narrow (about ¾ cm.) semi-transparent area. To the left of this, and rather forward and adjoining the coronal suture, was a triangular osseous island, also flanked by a transparent area.

The impressiones digitatæ and Pacchionian depressions were more pronounced on the left side.

On the occiput, where a trauma was sustained five years previously, no abnormal condition was observed.

The *pia* is moderately congested, and over the whole convexity is considerably thickened and opaque. The *brain* tissues appear normal. The thickness of the *parietal bones* varies greatly, especially along the sagittal suture.

Microscopical examination. The liver shows the usual lesions of advanced chronic congestion, with complete atrophy of cells at the centres of lobules, and slight fatty degeneration of cells at the peripheries of lobules.



The small nodules above described prove to be miliary adenomata. They are composed of thick cords of hypertrophic liver cells, usually containing more than one nucleus, and at some points showing considerable fatty infiltration. The larger adenomata are surrounded by a distinct fibrous capsule, within which effects of chronic congestion have not been felt, as the capillaries of the adenomata are not at all widened.[1] The cords of liver-cells surrounding the nodules are much compressed for a considerable distance. (See drawing.)

[1] In this respect our specimen differs from the majority of the few cases which have been described. In them the liver-cells were thoroughly compressed and destroyed by copious interstitial tissue, and new cells were developed through a compensatory process.

The lesions in the *lymph nodes* include:

1. A chronic inflammatory hyperplasia, with the production of new connective tissue, extensive exfoliation of endothelial cells, and atrophy of lymphoid cells.

2. An extreme dilatation of veins and capillaries, both within and without the nodes.

The inflammatory process has replaced considerable portions of the nodes by a tissue apparently composed of flattened and fusiform endothelial or connective cells, and entirely lacking in lymphoid cells. In these areas the capillaries are often dilated and gorged with blood. The lymph nodules are all quite small, and many appear to have been replaced by the above tissue. There is a slight deposit of anthracotic pigment in the fibrous tissue. The large neighboring veins are enormously dilated, and possibly increased in number so as to give an appearance not unlike that of a cavernous angioma. The venous stasis has likewise affected the vessels within the nodes, the medullary lymph cords being entirely absent and replaced by dilated blood-spaces or fibrous tissue. The condition of the lymph nodes indicates a process of older date than that of the changes in the liver.

The *mesentery* shows œdematous infiltration, and about some small vessels are collections of mononuclear and polynuclear cells.

The *spleen* shows the effects of chronic congestion equally marked with those of the liver. The sinuses are much dilated, and the pulp-cells contain a large deposit of blood-pigment.

The *intestinal wall* shows a light coating of fibrin with exfoliation of endothelial cells.

The *ovaries and Fallopian tubes* show venous congestion, but no other lesion.

Brain. The motor cortex of both sides was cut in thin slices and hardened in saturated watery solution of corrosive sublimate.

Sections stained by Nissl's method gave very unsatisfactory results, owing to advanced post-mortem changes. As nearly as could be judged, the motor areas of both sides were identical in appearances, including the numbers and configuration of the cells, and the character of chromaphilic bodies, and intracellular network. The pia of both sides was considerably thickened, that over the left motor area very much so.

Some capillaries were found containing colonies of cocci.

The ganglion cells of the cranial nerve nuclei (XII., X., IX., examined) showed no unusual appearances by Nissl's stain.

Bacteriological Examination. A portion of one of the superficial adenomata, including a section of the capsule of the liver, was inserted beneath the skin of a guinea-pig. Seven weeks later no inflammatory changes were to be found at the point of inoculation or in the adjoining lymph nodes.

A similar negative result followed the inoculation of another guinea-pig with a swab from the fluid and pus in the bottom of the drainage-tube. The fluid in the drainage-tube, smeared on cover-glasses, showed the presence of

numerous cocci in masses and short chains. Pure cultures of *staphylococcus pyogenes aureus* and of *streptococcus pyogenes*, of marked virulence, were secured by Dr. Charles Norris from this same fluid.

The fluid from the drainage-tube and sections of the intestinal wall, mesentery, liver, spleen, and lymph nodes were stained for tubercle bacilli, with a negative result.

The serosa of the inflamed intestines contained cocci in moderate numbers.

The spleen contained large numbers of minute colonies of cocci, and in the motor cerebral cortex and in a lymph node single colonies of cocci were observed.

Diagnosis. Chronic inflammatory hyperplasia of periportal and perihepatic lymph-nodes, of undetermined origin.

Partial compression of hepatic and portal veins.

Chronic congestion of liver and portal viscera. Multiple miliary adenomata of liver.

Ascites.

Peritonitis. Tubercle bacilli in the fluid.

Septicæmia.

Epicritical. From the pathological standpoint the obscure feature of the case is the relation of the swelling of the perihepatic lymph-nodes and the condition of the liver. In the absence of any other adequate cause it is necessary to conclude that the advanced chronic congestion of the liver was due to a compression of the hepatic vein from the enlarged lymph-node lying above and upon this vein. It then becomes necessary to discover a cause of this hyperplasia of the lymph-nodes, which, with the periportal nodes, were the only ones in the body thus affected. Several possibilities may be suggested. It is possible that the lymph-nodes were subjected to chronic irritation from intestinal toxæmia, in which case it is difficult to see how the mesenteric nodes could have escaped, as they did, a similar irritation and hyperplasia. The probability of a serious retrograde irritation from the thoracic chains must be regarded with caution, since the thoracic nodes were not enlarged, and the bronchial nodes showed only moderate pigmentation. The deposit of pigment in the perihepatic node was very slight and apparently quite insufficient to induce the lesion found in the node. Nevertheless such a possibility may be entertained.

Cholecystitis could have affected the periportal nodes, and suppurative lesions in this viscus commonly do so; but there were no

evidences, either gross or microscopical, of any disease of the gall-bladder. The considerable size of some of the adenomata ($\frac{1}{2}$ cm. in diameter) suggests that these may have been the primary lesions, and that the disordered condition of the hepatic circulation and function led to the hyperplasia of the lymph-nodes draining the organ. While there is no positive evidence on which to deny such a course of events, many recent studies of nodular hyperplasia of the liver tend to show that this lesion usually represents a regenerative tendency, well marked in liver-cells, to replace disordered or destroyed hepatic tissue by way of compensation. No other cause for liver congestion can be found in our case in heart, pleura, or lungs. Some extensive studies of this character have been contributed, as follows:

Flock, Ueber Hypertrophie und Neubildung der Lebersubstanz. *Deut. Arch. f. klin. Med.*, 1895, Bd. lv.

Marchand, Ueber Ausgang der acuten Leberatrophie in multiple knotige Hyperplasie. Ziegler's *Bact.*, 1895, Bd. xvii. p. 206.

Meder, Ueber acute Leberatrophie mit besonderer Berücksichtigung der dabei beobachteten Regenerationserscheinungen. Ziegler's *Beit.*, 1895, Bd. xvii. p. 143.

Therese, Des adenomes du foie. *L' Union Médicale*, 1895, No. 34.

Babes et Manicatide, Les proliferations des cellules hépatiques dans les differents affections du foie. *Ref. Cent. f. Path.*, etc., 1896, No. 23.

The very general distribution of the nodules in the present case, and the fact that they represented areas of apparently normal liver-cells—in the drawing (p. 500) the presence and absence of these cells in adjoining territory are beautifully represented—in which the effects of the chronic congestion were not at all noticeable seem to support strongly the view that these miliary adenomata were entirely the result of the advanced chronic congestion and atrophy of liver-cells which affected the entire organ.

As already stated, moreover, the lesion in the lymph-nodes appears to have been probably of older date than the adenomata of the liver.

Pressure on the hepatic vein having been once established, thereby obstructing the venous return from the nodes themselves, it is readily seen how the course of events should lead to exactly the condition found at autopsy.

The peritonitis and septicæmia which terminated the case may

properly be regarded as the natural and very frequent result of the sudden relief of pressure from the abdominal viscera, especially the intestines, allowing the passage of intestinal bacteria into the peritoneum and apparently, also, into the general circulation. The intestinal lumen contained a very abundant growth of cocci; the streptococcus and staphylococcus were isolated from the peritoneum, and bacterial thrombi composed of cocci were found in the spleen abundantly, and in the brain and lymph-nodes.

This consideration affords also a clue to the presence of tubercle bacilli in the serum contained in the abdominal cavity. They are transferred through the circulation, either of the blood or the lymph. The latter would start from lymph-nodes which were, however, nowhere affected, nor was there any organ or tissue infected with tuberculosis. Therefore the presence of tubercle bacilli can be explained by transmission through the blood-current only. Evidently they were only a complication, an incident. When the rapid effusion took place from the bloodvessels of the peritoneum and the intestine, where the bacilli happened to be located, without having given rise to local or general symptoms, the vessels were emptied of a thin serum—of 1011 specific gravity—with all its contents. The alkaline fluid in the abdominal cavity proved an excellent nutrient and a preservative for the bacilli.

The presence of bacilli in the ascitic fluid, in the absence as well of tuberculous deposits or degenerations anywhere, as of lesions in the intestine which could be charged with admitting the microbes into the circulation, is no longer an improbability, since it has been proven that surface lesions of a mucous membrane are not required to admit tubercle bacilli nor hiatuses in the epithelial covering, such as are possessed by the tonsils (Stoehr) in their normal state. It has been known for some time that initial lesions in the lungs, for instance, need not correspond with the localities of original affections, for pulmonary infiltrations will follow the subcutaneous injections of bacilli in distant places. Koch proved that lymph-bodies may become diseased without affections of their roots. Solid particles are swept through the lungs. The spores of saprophytes and of anthrax are so admitted. Whether this happens more readily in children, whose organs are less altered by the solid results of previous morbid processes, remains to be seen. Bollinger

and Heller demonstrated that tubercle virus may penetrate through intact tissue, and that we need not assume with Babes that cocci prepared the soil for absorption in every instance. The tubercle bacilli in the ascites of our case can, therefore, be explained in one of two ways. They were, before entering the abdominal cavity, contained either in the circulating blood or in the intestinal tract. The former is very improbable, for there were no miliary deposits anywhere. The presence of bacilli in the intestinal tract is explained by the facility of their admission with food. It is probable there are but few intestinal tracts but are harboring bacilli now and then. It is under favorable circumstances only that they are absorbed instead of being expelled. Such favorable circumstances are either the presence of local lesions or sudden changes of osmosis. The latter existed in our case.

The local cranial defects remain unexplained. If they had been found in the skull of a baby less than six or ten months old, they would certainly have been claimed as rhachitical. Craniotabes, however, after rhachitis gets well, seldom leaves behind any of these attenuated circumscribed spots. There was no symptom of rhachitis in the patient when an infant, except constipation. There was, moreover, no tumor, no thickening inside to explain a local absorption of such circumscribed character. I prefer to point to this instance of defective local bone development on some other than rhachitical basis.

It has been noticed that the pia and surface of the brain exhibited marked alterations from the normal, but none so circumscribed as to explain the localized spasmodic symptoms constituting the very picture of what we mean by the term Jacksonian epilepsy. The advisability of an operation undertaken to find and remove its local cause was often considered by the medical men who studied the case. It never was urged by anyone; it was advised against by some. Nothing that is positive can be learned from this feature of the case, except the necessity of care and caution and prudence.

A therapeutical remark will not be out of place. It has been seen that the bromides were sometimes required in such quantities as to interfere seriously with the general health of the patient.

When the doses of urethan recommended by Dr. Dana were given persistently the convulsive attacks ceased very soon, never to return, and there were no disagreeable symptoms attributable to the remedy.